The authors state that their procedure does not deform the tubercle bacilli, a fault which may be found with some of the other methods.

**Bacteriology of Whooping-Cough.**—DAVID J. DAVIS (*Journal of Infectious Diseases*, 1906, vol. iii. p. 1) carefully reviews the existing conceptions in regard to the etiological role of various micro-organisms in whooping-cough. Although there is great lack of unity in results arising chiefly from a diversity of methods, the author believes that practically all modern investigators have been dealing either in pure or in impure cultures with the influenza-like bacillus first described by Sprengler and later by Jochmann.

Davis made bacteriological examinations of the sputum of eleven cases of pertussis and was able to isolate an organism which he could not differentiate from the influenza bacillus in fifty-six cases, or in 92 per cent. The organism showed a low degree of virulence for laboratory animals, although a general bacteræmia and death were produced when large amounts of the organism were inoculated intraperitoneally. The virulence was increased when inoculated with non-virulent strains of staphylococcus pyogenes aureus and other organisms. This symbiotic effect the author considers quite important in the etiological role of the organism.

Inoculation of the throats and nasal cavities of monkeys had no effect. The organism obtained from pertussis cases was smeared on the tonsils and nasal mucosa of a healthy young man and a definite reaction followed. About forty-eight hours after inoculation the patient had chilly sensations, a cold sweat, some headache and weakness, and his temperature rose to 100.2° F. The febrile reaction continued for about two days. This was followed by a somewhat indefinite pharyngitis with cough for about four weeks. The cough was not, however, spasmodic and whooping did not occur. This experiment proves, the author thinks, that this influenza-like organism is at least not always a harmless saprophyte.

The throats of numbers of patients suffering usually with the diseases of childhood were examined, and a similar organism was found in from 18 to 80 per cent., varying with the disease. It was also isolated twice in cultures from the throats of twenty healthy medical students. The author believes that this bacillus cannot as yet be sufficiently differentiated from the influenza bacillus to warrant another name for it.

Although Davis considers that evidence at hand will not permit a definite statement for or against the specificity of the organism for whooping-cough, it is certainly very frequently found in the sputum of whooping-cough patients, especially during the spasmodic stage of the disease. It has been found several days before the whoops began and as long as six months after the disease. The work, together with that of Jochmann, Wollstein and others, certainly suggests that the final solution of the cause of whooping-cough will be found in the group of organisms to which the influenza bacillus belongs.

**Multiple Non-inflammatory Necrosis of the Liver.**—OERTEL (*Journal of Experimental Medicine*, 1906, vol. viii. p. 103) describes a peculiar type of necrosis of the liver which he has observed four times in two hundred and seventy-five autopsies. In the gross the liver appears pale, the markings are lost, and the surface presents circumscribed deep yellow or hemorrhagic spots and streaks. Microscopically, irregular areas sometimes forming streaks were seen, in which the liver cells were destroyed. The necrosis was general. The distribution of the single areas varied, sometimes being found about the central vein of the lobule, sometimes about the periphery. In these foci, which were often circumscribed, but at times had no definite margins, the liver cells presented various grades of necrosis without inflammatory reaction. The outlines of the cells were well preserved. The nucleus showed all stages of fading to complete disappearance. The cells contained many bile granules and fat droplets. In the areas where the process was more advanced the liver cells had undergone complete dissolution, leaving only a shadow or an empty space with the surrounding reticulum. In all the cases there was more or less portal sclerosis. The condition differs altogether from the ordinary parenchymatous degeneration or coagulation necrosis of cells. The author excludes a postmortem change and suggests that the necrosis is caused by an autolysis of the liver cells; and since the lesion cannot be classified with the known forms of necrosis of the liver, it must be considered as a new type hitherto undescribed.

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**Changes in the Pancreas in Cirrhosis of the Liver.**—LANDO (*Zeit. f. Heilk.*, 1906, Bd. xxvii. p. 1) has studied the pancreas in twenty-three cases of cirrhosis of the liver. In most instances the cirrhosis was of atrophic type and in fifteen cases there was a history of alcoholism. Diabetes was present in three instances. The pancreas in all cases showed more or less overgrowth of connective tissue which in twelve cases was about proportionate to the cirrhosis of the liver. The pancreatic cirrhosis was at times entirely intralobular and confined to the interacinar connective tissue; at other times the increase was principally in the interlobular connective tissue, though in this type there always existed some increase in the intralobular connective tissue too. The head and tail of the pancreas seemed to be affected first and exhibited the most extensive changes. In a case of pigmentary cirrhosis of the liver there was also marked pigmentation of the pancreas. The islands of Langerhans presented usually some alterations which, however, except in the cases of diabetes, were marked neither in degree nor extent and consisted in slight thickening of the capsule or vessels and degenerative changes of the epithelium. In two of the cases of diabetes there was hyaline degeneration. Increase or thickening of the elastic tissues of the organ was often noted. The weight of the pancreas varied from 32 to 150 grams. The form of the cirrhosis, whether atrophic or hypertrophic, made no difference in the microscopic picture of the changes in the pancreas. Two principal causes for the lesions in the pancreas are discussed. It is possible, first, that the changes in the pancreas may be the sequence of a chronic passive congestion following the cirrhosis of the liver; or, secondly, the etiological factor in the production of the cirrhosis of the liver may also be the cause of the changes in the pancreas. In most of the cases the author adopts the latter view, regarding alcohol as the etiological factor. In a few instances he believes that the pancreatic lesion followed directly from the cirrhosis of the liver.